

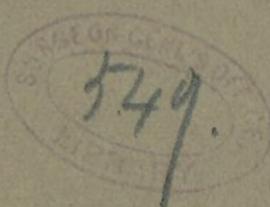
WRIGHT (J.)

TUBERCULOSIS OF THE  
UPPER AIR-PASSAGES.

*AETIOLOGY.*

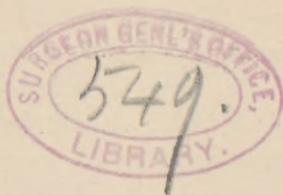
BY  
JONATHAN WRIGHT, M. D.,  
BROOKLYN.

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## TUBERCULOSIS OF THE UPPER AIR-PASSAGES.\*

### AETIOLOGY.

BY JONATHAN WRIGHT, M. D.,  
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I HAVE been asked to discuss that part of the subject of tuberculosis of the upper air-passages included under the heading of aetiology. This can not be successfully done without considering the aetiology of tuberculosis, to some extent, in its general aspect. That is a broad field which reaches out in all directions. Much of it is unexplored. Perhaps some of it is not accessible at present. Eight or ten years ago it seemed as though that tiny organism, the tubercle bacillus, filled the field completely.

Of late I have been looking back to my bacteriological days and wondering a little that we now hear no more of the three "postulates of Koch" which were so familiar to us then. They have not been heard of for so long that I have almost forgotten exactly how they were worded. As I remember them, however, they were that no micro-or-

\* Read before the American Laryngological Association at its seventeenth annual congress.

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ganism could be considered an ætiological factor in any disease unless—

1. It was always present in that disease.
2. It was never found in man without the disease.
3. Inoculation with it would always cause the disease.

These proud legends were inscribed on the banner of the “high contagionists.” This is not the place to speak of the history of the researches into the activities of other disease germs than the tubercle bacillus, but suffice it to say that in that history also may be observed the downfall of the postulates. As regards the tubercle bacillus, it is now always present in tubercle, somewhat on account of similar tactics to those used by Mohammed with the mountain. The old clinicians had been in the habit of calling many things tubercle, but the new bacteriologists ruled out all those pathological structures not due to the action of the bacillus. And this has proved a great gain in clinical classification, but, like all classifications in medicine, it has wrought some evil in crystallizing a certain order of mental process into a form of intellectual non-receptivity which renders further advance in individual cases impossible.

As to the second postulate, it has disappeared so completely that hardly any traces of it can be found in contemporary medical thought. The tubercle bacillus is found not only dormant in animal tissues, entangled in the meshes of lymph glands and in unsuspected foci in the lungs, but it has lately been found, as has the diphtheria bacillus, in the upper air-passages of healthy people.

It must have occurred to many others as well as to myself that the principal reason it has not been found before, and is not now found more frequently, is the difficulty of technique attending its demonstration. This suspicion is strengthened by a very suggestive bit of medical news that

comes from Paris by way of the *Lancet* of May 11, 1895 (p. 1220):

*Latent Tuberculosis of the Tonsils.*—Professor Dieulafoy calls attention to a torpid variety of pharyngeal tuberculosis the favorite seat of which is the adenoid tissue of the nasopharynx. This tuberculosis manifests its presence by an exuberant growth of the lymphoid organs of that region—in other words, by hypertrophy of one or more of the palatine and pharyngeal tonsils. This view of Professor Dieulafoy would, if confirmed, lead us to regard hypertrophy of the tonsils and adenoid growths as, in many instances, cases of tuberculous overgrowth of adenoid tissue. He bases this belief on the results of inoculations practised on guinea-pigs of fragments of enlarged tonsils and adenoid vegetations. Of sixty animals thus inoculated with tonsil tissue, eight, or thirteen per cent., succumbed to generalized tuberculosis, while of thirty-five inoculated with adenoid tissue, seven, or twenty per cent., became tuberculous. In all the persons who furnished the material for inoculation (enlarged tonsils and adenoid growths) the pharyngeal tuberculosis was primary and not consecutive to the pulmonary variety. It is to be supposed that the young subjects who have enlarged tonsils, etc., provide a favorable soil for the growth of the bacillus of Koch, which finds access to the adenoid culture medium either with the food, milk especially, or with the air respired (sojourn in a bacillary atmosphere). Professor Strauss, of Paris, has, indeed, demonstrated the presence of virulent tubercle bacilli in the nasal cavities of individuals habitually breathing the same air as phthisical patients. An open wound is not necessary for penetration, since the bacilli can find an entrance through the epithelium. In some instances the bacilli present in the adenoid tissue are, after a sojourn of months or years, destroyed by phagocytosis, which determines an indurating, fibrous process in the tonsil. In other cases, however, the bacillus finds its way into the lymphatic vessels, and enlarged submaxillary and cervical glands are the result. This lymphatic infection is often

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started by the occurrence of measles, scarlet fever, whooping cough, etc. This glandular tuberculosis may, in its turn, remain local, and finally end in recovery ; but in other instances rapid generalization may result. The third stage of tonsillitic tuberculosis is the spread of the process to the lungs, the bacillus reaching those organs from the cervical glands via the lymphatics, thoracic duct, and the right heart. In the course of the discussion raised by this most interesting communication M. Chauveau stated that in animals fed on tuberculous matter infection may take place by inoculation of the adenoid tissue of the base of the tongue and the isthmus of the pharynx, this inoculation being proved by the swelling of the cervical and submaxillary glands. Sometimes a minute erosion explains their inoculation, but often the surface was found intact.

I have read this extract in its entirety, but it is too early, without having seen the original paper of Dr. Dieulafoy, to make any comment on the subject beyond saying that, although Lermoyez has reported two cases of tuberculosis following operations for post-nasal adenoids, we are not accustomed to expect any such result either from an adenotomy or from an amygdalotomy. I only quote it here to show how far we are from the second postulate mentioned above.

Now in regard to the third article in the early bacteriological creed. It has been invalidated somewhat indirectly by the general trend of facts recently ascertained such as I have just instanced. It is doubtless true that a Pravaz syringeful of broth swarming with tubercle bacilli will kill *any* guinea-pig. It is very likely true that it will kill *any man*, though direct evidence on that point is lacking, but clinically a Pravaz syringe thus used does not enter into the aetiology of tuberculosis.

I need not pursue ancient history further. Indeed, some of you are doubtless already saying that I am setting

up men of straw for the pleasure of knocking them down. Until lately, however, they were not thought men of straw, and I have taken these early postulates as an excuse to place before you a little of the evidence which we have clinically and experimentally, that the tubercle bacillus, although being the "*sine qua non*" of tuberculosis, is after all practically, especially from a prophylactic or hygienic point of view, a minor element in its multitudinous aetiological factors. I think we are gradually approaching a belief that every one at some time in his life comes in contact with such a dose of the tubercle bacillus that, were the other aetiological factors also present, he would die of tuberculosis. We know that one in seven deaths are due to tuberculosis. The autopsy table has shown that in one in two or three of all the cases examined there is evidence discoverable of the active or the conquered onslaughts of the bacillus. Now there *must* be a good many other cases in which the tubercle bacillus has perished without leaving a trace behind him—on the skirmish line as it were.

Cornet showed a good while ago the high rate of mortality among those confined in prisons and workshops from tuberculosis, and he reasoned that it was because of the contact with the tubercle bacillus.\* I need not follow the

\* Since this was written, I have lately seen that Cornet has reiterated his belief, though not quite so emphatically. He said in a paper read before one of the German medical societies that the mortality from phthisis had fallen greatly since the institution of precautions recommended by him against infection from tuberculous sputum. In the discussion which followed, Baer rather spoiled the force of this remark by expressing a doubt as to whether the mortality from phthisis had diminished on account of these precautions or because of the general reform in furnishing the prisoners with better air, food, and exercise. For a very strong plea for the side of the question opposing the views I have expressed here, see Cornet's paper and the discussion in the *Berl. klin. Woch.*, May 20, 1895.

history of clinical and experimental investigations which make us now believe that the high mortality is due not to the bacillus, but to the prison and workshop. It has been conclusively shown that no higher mortality exists among the consorts of tubercular patients than among other husbands and wives, but the page of every clinical history book will show a different story where the children of such patients are concerned. Is the death-rate of tuberculosis strikingly high among doctors? Is it the impression of the members of this association that more than one in seven of our *confrères* die of tuberculosis?\* In the hospitals and dispensaries and in our offices we meet the tubercle bacillus face to face every day. It is gradually becoming known that a very large proportion of all dairy cows, especially of the Alderney breed, are tubercular. It is the children of the well-to-do, if not of the rich, that are brought up on this milk. The poor suckle their children themselves. It is hardly necessary to ask among which class we find the largest proportion of tubercular children. This fact has been noted for years, and so far as I have observed has never been satisfactorily explained by the "high contagionists."

Experimentally Prudden has shown that he can produce a lesion identical with tubercle without the action of the *living* tubercle bacillus, and a French author has said *inversely*: "The bacillus is not enough, we must have the characteristic reactional lesion—the tubercle." As clinicians we may say, and we should say it boldly, that neither the tubercle nor the tubercle bacillus, nor both together, are enough. The tubercle bacillus is at one end of the chain and the tubercle is on the autopsy table at the other end of

\* Dr. Kortright, in the *Brooklyn Medical Journal*, June, 1895, says that statistics prove that the doctors do not average as high a mortality from phthisis as other classes in the community.

it. Tuberculosis to us does not mean either of these. It means to us both of these, plus the vital energy of the bacillus on the one hand and that of the resisting human organism on the other.

We group all the other factors in the general aetiology of tuberculosis under the head of "predisposition," divided into such vague terms as low vitality, heredity, scrofula. And apropos of scrofula, before leaving the subject of general aetiology for that of the special aetiology of tubercular disease of the nose and throat, I desire to quote some words of Ziegler (*Allgemeine Pathologie*, etc., 8te Auflage, Band i, S. 614) :

"According to our experience, the disposition to tuberculosis is in the human race a greatly varying one, since only a part are predisposed to it. According to current views, scrofula—*i. e.*, a sickly condition of the organism, which is revealed by a tendency to certain disturbances of nutrition of the skin, of the mucous membranes, of the joints, of the bones, and of the lymph nodes—predisposes to it. It may here be remarked that many of the manifestations which are ascribed to *scrofula* are really already manifestations of a tubercular disease."

Of course, in these few remarks I have not pretended even to touch on all the points in the general aetiology of tuberculosis, but have only hinted at some of them, and we must now consider those special influences which determine its primary and especially its secondary location in the upper air-passages.

There are certain facts which seem clearly to indicate that there exists in the nose and throat some special local resisting power exerted against the entrance of the tubercle bacillus, or that there exists some special local annihilating influence upon the bacillus after it has gained an entrance into these mucous membranes. If we are not disposed to

deny the possibility of a really primary occurrence of tuberculosis in the larynx, we are certainly not in a position to deny that even its apparent occurrence is an extremely rare clinical phenomenon. Supposing that the inspired air is the bearer of tubercular infection to the lungs, the conclusion inevitably follows that the nose and throat, as compared to the lungs, must possess a more complete protection, for elsewhere I have shown experimentally that the former retain from the inspired air all but a fraction of the floating germs before they reach the bronchi. Were it not for some protecting factor, primary laryngeal tuberculosis should be at least as common an occurrence as primary pulmonary tuberculosis.

Krückman \* has lately shown, by a large number of very carefully conducted examinations, that in adults the tonsils and cervical glands are usually infected by the tubercle bacillus *after* the lungs are the seat of disease.

On the other hand, in children, he seems to believe, and we know that clinical evidence goes to show, that the cervical lymphatics are usually affected before the pulmonary portachyma. Supposing, however, that the tubercular infection is carried by the lymphatics in both adults and children, we reach the same conclusion of comparative laryngeal immunity when we remember how extremely rare tubercular laryngitis is in children.

Now what are these protective factors in the upper air-passages? So far as lymphatic channels of infection are concerned in the larynx, we may advance the explanation of the scanty anastomosis of the internal and external vessels. We have a clinical evidence of this in the late stage at which enlarged cervical lymphatics are discoverable in

\* Krückman. *Virchow's Archiv*, No. 138, Heft 3, p. 534. I also desire to call attention to the paper published by Sims Woodhead in *The Lancet*, October 27, 1894.

the cases of laryngeal cancer, but this will not explain the pharyngeal immunity.

If Dieulafoy's recent observations are reliable, we may explain by them the origin of some of the cases of tubercular meningitis in children, for there is direct and abundant communication between the naso-pharyngeal and the intracranial lymphatics, but these observations, if accurate, throw no light on the cause of comparative immunity of the nose and throat in children to recognizable tubercular lesions, but on the contrary would make it more apparent, for I imagine it will be shown that lymphoid tissue is not the product but the lurking place of the tubercle bacillus.

The same reasoning applies when we remember the fact that only fifteen to thirty per cent.\* of all cases of pulmonary tuberculosis, even at autopsy, are seen to be also victims of laryngeal tuberculosis. As said before, all cases should present laryngeal lesions, were there not some protection against the virulent cultures of the bacillus in the sputum which bathes the larynx.

I do not propose to go into the history of the literature

\* Heinze is the only author who goes as high as fifty-one per cent. It must be remembered that these are statistics from the autopsy table, and these must differ materially from what is observed laryngoscopically for three reasons: Firstly, and chiefly, comparatively few persons dying of phthisis have ever had a laryngoscopic examination. Many tubercular lesions of the nose and throat exist without symptoms referable to them. Secondly, many tubercular lesions may escape observation even in the most thorough rhinoscopic and laryngoscopic examination. Thirdly, many tubercular ulcers appear in the upper air-passages only when the patients are "*in extremis*."

Through the kindness of Dr. Arrowsmith and of the other attending physicians, I have been able to examine twenty-five patients in the Brooklyn Home for Consumptives, all of them suffering from the lesions of the second and third stages of pulmonary phthisis. In only four of these, or sixteen per cent., were tubercular lesions found in the nose and throat.

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of laryngeal tubercular infection. You are all familiar with the old theory of Louis as to the corrosive action of the sputum. It occurs to me that the chief objection to Louis's doctrine, as he meant it, is that no one has yet shown that sputum from tubercular lungs possesses any chemical erosive properties. You know that Eugen Fraenkel has demonstrated the tubercle bacillus in the surface epithelium and in the superficial layers of the mucous membrane. This has not been confirmed by many of the workers at the problem, but lately has been corroborated by the investigations of Mr. Lake, published in the April number of the *American Journal of the Medical Sciences* for 1895. He says, however, that the pyogenic cocci first make a breach by getting in between the epithelial cells and, multiplying there, form little abscesses which result in superficial erosions through which the tubercle bacillus itself enters, it having been shown that the bacillus itself is unable to produce abscesses. It is not necessary here to discuss Friederich's idea that the pneumogastric, in some cases of pulmonary phthisis, by becoming affected produces a trophic lesion in the larynx which allows the tubercle bacillus to enter.

I must, however, refer to an excellent paper by Thost,\* who claims that the bacillus frequently enters by way of the glands, in whose epithelium he claims that tubercle frequently develops. This, however, is strenuously denied by Fraenkel. It is well to remind you here that Virchow long ago cited the larynx as the situation where typical tubercle structure could best be studied microscopically.

For several years I have been in the habit, when opportunity offered, of making sections transversely through the trachea and larynx of stillborn infants or of those dying shortly after birth. I had frequently noticed that the epi-

\* *Monatschr. f. Ohrenheilk.*, No. 2, February, 1893.

thelial layer in these transverse sections had a wavy outline—that it was thrown into folds; it seemed too redundant as a lining for the tube. In the larynx this is especially noticeable in the interarytænoid space. At first I supposed these folds were due to the shrinking from prolonged soaking in alcohol. Lately, however, I have examined specimens prepared by preliminary fixing in four per cent. formalin, and the same phenomenon is observable, as you see in this drawing of a section from just below the vocal cords by camera lucida and a low power (Zeiss A) objective (Fig. 1).

If you will examine the beautiful photographs made by Prof. B. Fraenkel \* of similar sections in adults, you will note the same appearance. Both in adults and infants, and also in animals (I have sections of pigs' and calves' and kittens' larynges), it will be seen that the epithelial and subepithelial layers are connected by very loose areolar tissue with the underlying firmer fibro-elastic connective tissue and perichondrium of the air tube. Now this evidently subserves a very important physiological function, and one which has a direct bearing on our subject. When a child screams, when a stentorian street hawker shouts his wares, when a consumptive has a paroxysm of coughing, the air tube is put on the stretch, the fibro-elastic elements may dilate by the shortening of the trachea as the thorax is heaved up, but the epithelial layer would suffer constant damage were it a "tight-fit" lining. The greater part of the epithelium is columnar. These long, narrow cells lie side by side, with their ends pointing outward toward the enemy. Were there no folds to be smoothed out like an accordion pleat, those cells at some one or more points must be dragged apart whenever the air tube should be violently distended.

\* B. Fraenkel. *Archiv für Laryngologie*, Band i, Heft 1 and 2.

In adults these folds are less marked than in children ; the areolar tissue binds the epithelial layers more firmly to the walls. Every attack of laryngitis tends to damage this physiological arrangement by stiffening the subepithelial

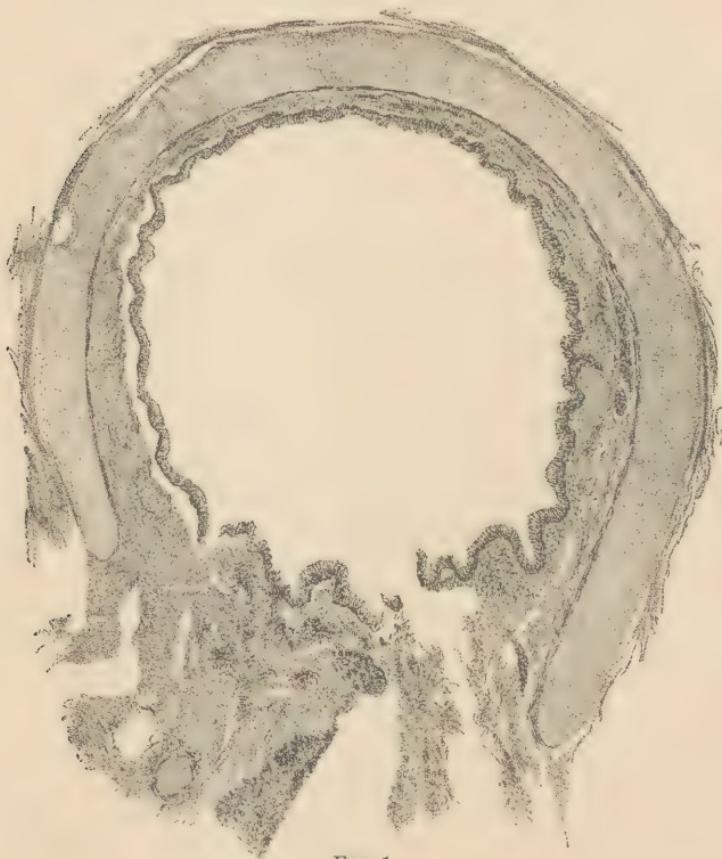


FIG. 1.

structure and by hyperplasia of the epithelial cells. Much more must this be the case in chronic inflammations, where we also find some metamorphosis of the columnar into the squamous type of epithelium. This latter is especially

seen in phthisis on the posterior laryngeal wall. E. Fraenkel states that the base of the arytaenoid cartilages at the internal and opposing faces of the vocal processes, where rubbing occurs during phonation, is the most frequent site of beginning laryngeal tuberculosis.

This may possibly be true of ulceration, but my clinical experience disposes me to think it is the interarytaenoid space. That situation at least is surely the place in which we first see that heaping up of pearly epithelium which is so significant to the experienced laryngologist.

After this study of physiological and pathological processes we are prepared to admit many of the causes to which are usually ascribed the supervention of laryngeal upon pulmonary tuberculosis--low vitality, the paroxysmal cough, the mechanical rasping of the sputum against the laryngeal walls, especially at the posterior commissure, repeated attacks of acute laryngitis or the presence of the results of chronic laryngitis, the overuse of the voice, exposure to cold and dusty winds, etc.

Tubercular ulceration of the pharynx is usually seen only in the very last stages of pulmonary and laryngeal phthisis, when the systemic and local vitality is at a low ebb, or in acute miliary tuberculosis when we have apparently some sudden weakening of the special factor which protects the system ordinarily from the ravages of the tubercle bacillus. Most of the cases of nasal tuberculosis which I have seen have been due to this low vitality at the end of pulmonary phthisis, and those cases of tubercular tumor which I have read of and not seen are apparently due to the abrasion of the finger-nail or to some other traumatism, as they are mostly reported as occurring anteriorly on the *sæptum* or on the anterior ends of the turbinate bones.

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There is no more time at my disposal to enter into a discussion of the question of the spread of tuberculosis by means of the lymphatics and blood-vessels. While it seems apparent that the method of infection is usually from the external surface through a gap in the epithelium, it can not be denied that the larynx is theoretically just as apt to be affected (barring the scantiness of the lymphatics) by means of the lymph and blood vessels as are the knee and hip joints; and practically there are many clinical and morphological facts which go to sustain its actual occurrence in many cases.





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